substance use. Although many adolescents who smoke do not become regular users of other drugs, there are typically a concurrent correlation between smoking and other types of drug use (Hays, Stacy, DiMatteo 1984; Single, Kandel, Faust 1974; Revell, Warburton, Wesnes 1986) and a statistical relationship between early cigarette smoking and subsequent use of hard liquor and marijuana (Kandel 1975; Donovan and Jessor 1983). There is no direct evidence linking multiple drug use to mood regulation effects, but it has been shown that negative life events are a risk factor not only for cigarette smoking, but also for several types of other drug use (Bruns and Geist 1984; Kellam, Brown, Fleming 1982; Newcomb and Harlow 1986).

For interpretation of data on stress and smoking in adolescents, the primary methodological issue concerns a possible third confounding variable. It may be that high levels of subjective stress are most prevalent among adolescents who have difficulty adjusting to school and family because of underlying psychopathology (Depue and Monroe 1986) and who identify with the values of a deviant lifestyle that includes substance use and delinquent behavior (Jessor and Jessor 1977). The current evidence argues against this interpretation; some data show that stress-smoking correlations remain significant with control for variables such as risk-taking, perceived control, and self-esteem (Hirschman, Leventhal, Glynn 1984; Newcomb and Harlow 1986; Wills 1985), and it has been shown that negative life events that could not be self-caused by adolescents show an independent predictive relationship to smoking (Wills 1986). The current evidence, however, is minimal and does not clearly rule out the alternative interpretation. At present it can be concluded that subjective stress may be a risk factor for adolescent smoking.

Stress and Cigarette Consumption

In considering evidence on affective factors and cigarette consumption among regular users, both epidemiological and laboratory data are available. Designs in the epidemiological studies are relatively weak because studies are largely cross-sectional, making causal interpretation difficult. When longitudinal data are available, the followup periods are rather short (approximately 1 year) in relation to the probable time course of stress-smoking relationships in adult populations. The following section presents the epidemiological evidence and laboratory studies of stress and smoking.

A large body of personality research has linked measures in the category of "neuroticism" to cigarette smoking among adult populations (Kozlowski 1979). These measures, which include scales of nervousness, emotionality, and anxiety, are conceptually similar to the concept of negative affectivity as defined by Watson and Clark (1984); that is, the tendency to perceive and experience negative

affect. Theoretically, this is the most relevant construct for examining links between affective factors and smoking. Of the 50 studies reviewed by Kozlowski (1979), half showed a significant relationship between neuroticism and smoking. Three studies in this literature showed the relationship between neuroticism and smoking to be more characteristic of females than males (Cherry and Kiernan 1976; Clausen 1968; Waters 1971). These studies were mostly cross-sectional, making inferences of causality problematic because of the possibility that smoking caused feelings of anxiety and depression. Also, Cherry and Kiernan (1976) analyzed longitudinal data and found that neuroticism predicted initiation of smoking by women but neuroticism predicted decreased likelihood of quitting by men. One prospective study (Seltzer and Oechsli 1985) related personality measures obtained at age 10 to smoking status at age 16 in a sample of 1,127 subjects from health maintenance organizations in the Oakland, California, area. The prospective analyses showed that measures of anger, restless sleep, and Type A personality were significantly related to onset of smoking. These analyses were performed with control for parental socioeconomic status and smoking. Measures of neuroticism and anxiety did not discriminate smokers in these analyses.

In the laboratory, smokers tend to smoke more during stressful situations (Epstein and Collins 1977; Rose, Ananda, Jarvik 1983; Schachter et al. 1977). Individuals attempting to quit smoking tend to experience relapses into a state of continued smoking during stressful situations (Shiffman 1986). Such findings are consistent with the self-reported claims of smokers that they smoke in order to reduce stress-induced negative affect. However, there is no convincing research evidence to indicate whether smoking actually reduces stress. It may be that smoking reduces stress relative to smoking deprivation or that smoking increases during stress without attenuating it.

It has been suggested that smokers smoke as a technique to deal with stress (Wills 1985). If smoking is indeed used as a coping mechanism, individuals with poor coping skills and/or with high degrees of chronic stress would be expected to have a higher prevalence of smoking. Three prospective studies have found associations between anxious, aggressive, and generally neurotic personality traits in childhood and the tendency toward smoking later in life (Cherry and Kiernan 1976; Lerner and Vicary 1984; Seltzer and Oechsli 1985). Cross-sectional surveys have repeatedly supported these findings, showing that neurotic, depressed, angry, and rebellious individuals are more likely to smoke compared with more emotionally stable individuals (Spielberger 1986). Ninety percent or more of alcoholics smoke (Istvan and Matarazzo 1984) compared with about 30 percent of the general adult non-alcoholic population in the

United States. Individuals who commit suicide are much more likely to be smokers (Cederlof, Friberg, Lundman 1977; Doll and Peto 1976). It has been argued that individuals with personality disturbances and related psychological problems may, in some cases, be using nicotine as a form of self-medication (Brown 1973; Warburton, Wesnes, Revell 1983). It has also been noted that the symptoms of nicotine withdrawal syndrome are very similar to those of clinical depression (Gilbert and Welser, in press). Emotional and psychological disorders with high incidences of tobacco consumption are characterized by high degrees of negative affect, and it seems likely that, like other tobacco consumers, individuals with such disorders use tobacco as a means of coping with negative affect and stress.

Recent studies have used measures more directly linked to the experience of stress. In a survey of a sample of 505 Navy men on amphibious assault ships, Burr (1984) employed a 19-item measure indexing perceived stress from the domains of job, organization, and family and related the stress scales to a single item about smoking status. Results showed that two scales from the stress measure, indexing Role Conflict and Family Strain, were significant discriminators of smokers and nonsmokers in this sample. These results are cross-sectional, but were obtained in a multivariate analysis that included a measure of locus of control. Similar results were found in a cross-sectional study by Tagliacozzo and Vaughn (1982) in a sample of 448 hospital nurses, using a 26-item inventory of job-related stress. In this study, the stress-smoking relationship was found primarily among respondents who were younger (<28 years) and single. Billings and Moos (1983) studied a community sample of 608 adult respondents in the San Francisco area and found that heavy smokers differed from nonsmokers in showing higher levels of anxiety/depression symptoms and negative life events (during the previous year) in the areas of work strain and family illness. Correlations between stressors and amount of smoking were found primarily for heavy smokers, not for light smokers in this population. These data are consistent with findings from a community sample of 938 adults in New Haven (Lindenthal, Myers, Pepper 1972). This study found that a high level of negative events (during the previous year) was related to increased rates of smoking, with some data suggesting that this effect occurred primarily among persons scoring high on psychological impairment as measured by the Gurin Index. In this study the relationship between stress and smoking held with control for sex, race, age, marital status, and social class.

Only two studies have examined smoking and stress at more than one time point. Conway and associates (1981) studied a sample of 34 Navy officers in a training setting. Data were obtained on stressors and smoking for 14 study days over an 8-month period. The days were categorized by independent raters for stress level; additionally,

subjects made a daily rating on an eight-item scale of mood and subjective stress. Results showed that rates of smoking were significantly correlated with both the daily subjective stress measures and with the objective categorization of days for stress level. Items on perceived stress, anger, fatigue, and fear were significantly related to smoking in the overall sample, but an item on depression was not significantly correlated with smoking. Within-subject analyses of stress-smoking relationships indicated that the significant overall correlations were apparently due to a small number of individuals, but there were no data presented to discriminate these more reactive individuals from other members of the sample. A prospective study by Aneshensel and Huba (1983) was based on longitudinal data from four time periods with a community sample of 742 adult respondents in the Los Angeles area. Data on cigarette smoking, scored on a 1-to-5 scale, were obtained at baseline and at a 1-year followup interval. Results showed that a baseline measure of depression was not related to smoking either concurrently or over the 1-year interval.

The field studies are, for the most part, ambiguous with respect to causal interpretation. This difficulty is alleviated in laboratory studies in which subjects are randomly assigned to conditions and predictor variables are experimentally manipulated. Several studies of stress and smoking in laboratory settings have consistently found that stress increases rates of smoking. The stressors manipulated include threat of electric shock (Schachter et al. 1977), noise (Cherek 1985; Golding and Mangan 1982), and performance anxiety (Rose, Ananda, Jarvik 1983). These latter researchers also employed a concentration task and found that smoking increased in both the anxiety and concentration conditions, compared with a control condition. One study, using a public speaking manipulation, failed to find a significant effect of stress on smoking (Glad and Adesso 1976).

Based on epidemiological and laboratory research, it can be concluded that stress increases the rate of smoking among regular smokers. The convergence of results from cross-sectional, retrospective, and repeated-measures studies, in combination with findings from laboratory research, supports the interpretation of a causal relationship. There is some evidence suggesting that life stress has a greater impact among heavy smokers and among persons scoring high on negative-affect measures, but evidence on individual differences in this literature is minimal. The psychological mechanisms linking stress to increased smoking have not been clearly demonstrated (Leventhal and Cleary 1980; Schachter, Silverstein, Perlick 1977; Pomerleau and Pomerleau 1984). It may be that smoking attenuates stress (e.g., by regulating mood), that smoking increases during stress but does not attenuate it, or that smoking during stress is experienced as less stressful only when compared with smoking

deprivation during stress. Some laboratory studies and substantial theoretical speculations have addressed these issues and are discussed below.

Do Smoking and Nicotine Reduce Stress and Improve Mood?

There is evidence that smoking is perceived as helpful for coping with stress and dysphoric mood. A further question is whether smoking actually reduces stress or improves mood. In epidemiological studies, this question has not been directly addressed, a major limitation in the literature. There are some laboratory studies that bear on this question. This Section summarizes experimental findings concerning the effects of smoking and nicotine on stress and affect modulation.

Self-Reported Stress Reduction and Affect Modulation

Smoking-deprived smokers usually report more negative affect than do smokers who are allowed to smoke if the setting is one which tends to produce mild-to-moderate negative affect. Compared with those deprived for an hour or more, individuals allowed to smoke report less anxiety (Gilbert and Spielberger 1987; Heimstra 1973; Pomerleau, Turk, Fertig 1984; Jarvik et al., in press) as well as less anger and irritation (Cetta 1977; Heimstra 1973; Neetz 1979) during performance of a variety of slightly stressful tasks. Tobacco deprivation is also associated with self-reports of decreased alertness, lessened mental efficiency, and increased boredom during a variety of cognitive tasks (Frankenhaeuser et al. 1971; Heimstra 1973).

Experimental research suggests that nicotine is the most important, and possibly the essential, component of the affect-modulating properties of tobacco use (Gilbert and Welser, in press; Pomerleau and Pomerleau 1984). For example, studies comparing the effects of nicotine-containing gum with no-nicotine placebo gum report that nicotine reduces negative affect in nicotine-deprived habitual smokers (Hughes et al. 1984; Jarvis et al. 1982; West et al. 1984). In addition, habitual smokers assigned to smoke cigarettes of normal nicotine yield report less negative affect than those who smoke very-low-nicotine-yield cigarettes (Gilbert 1985; Perlick 1977).

However, a number of studies have not observed reduced negative affect due to smoking high- versus low-nicotine-yield cigarettes (Bowen 1969; Dubren 1975; Fleming and Lombardo 1987; Gilbert and Hagen 1980; Gilbert 1985; Hatch, Bierner, Fisher 1983). Gilbert and Welser (in press) suggest that these studies included inadequate periods of tobacco deprivation and excessively rapid smoking of multiple cigarettes (probably producing nicotine toxicity). Degree and type of stress to which subjects are exposed may also influence outcomes. There is evidence suggesting that nicotine has stress-

attenuating effects when stressor stimuli are mild or moderate, distal (anticipatory), and ambiguous, but fails to have such effects when stressors are brief, proximal, and/or intense (Gilbert and Welser, in press). More research is needed to evaluate these possibilities.

Behavioral Indices of Stress Reduction and Affect Modulation

A small number of studies that used behavioral indices of affect support the hypothesis that nicotine can reduce negative affect. Several studies report that smoking, or smoking a high-nicotine relative to a low-nicotine cigarette, is associated with reduced aggression (Cherek 1981; Schechter and Rand 1974). However, Jones and Leiser (1976) found no such effects on aggressive behavior by using similar procedures. In addition, without nonsmokers as controls, it is impossible to know whether the differences that were reported between conditions resulted from nicotine administration or nicotine deprivation.

Hughes and colleagues (1984) asked spouses to provide daily ratings of the subjects' behavioral indications of mood. These subjects had abruptly quit smoking and were randomly assigned to chew placebo gum or gum containing nicotine. Subjects who chewed the placebo gum were rated by their spouses as exhibiting significantly more anger and tension after quitting smoking, while those who chewed nicotine polacrilex gum showed little change in these emotional states. Thus, it appears that the nicotine provided by the gum replaced the nicotine previously obtained by smoking, so that there was little change in mood. However, it also appears that nicotine deprivation resulted in the tension and anger and that nicotine did not reduce these variables below baseline values.

Several studies have used pain thresholds as dependent variables in assessing the effects of smoking and nicotine on anxiety. Two studies that tested the effects of smoking cigarettes of different nicotine yield on electric shock endurance report elevated endurance thresholds in subjects who smoked relative to those who did not and in the high-nicotine-cigarette conditions relative to the low-nicotine-cigarette conditions (Nesbitt 1969; Silverstein 1982). The increased willingness to endure electric shock by individuals in the smoking and high-nicotine conditions was interpreted by these investigators and others (Schachter 1973) as indicating that nicotine reduces the anxiety associated with the electric shock. Other studies used the length of time that individuals are willing to endure pain associated with immersion of a hand or foot in ice water (the cold-pressor test) as an indicator of anxiety. These studies also showed that smoking and another means of nicotine administration (snuff) increase endurance in this test. However, the anxiolytic interpretation of increased pain thresholds has been questioned (Gilbert 1979),

because of the observation that in some situations nicotine has been reported to increase detection thresholds for tactile (including electrical) stimuli. It may be that nicotine reduces sensitivity to pain directly, rather than via reduction of anxiety. Several studies have failed to find increased shock endurance thresholds associated with smoking (Jarvik et al., in press; Milgrom-Friedman, Penman, Meares 1983; Shiffman and Jarvik 1984). In addition, it is unclear whether smoking and nicotine reduced these operational estimates of stress or whether smoking deprivation increased them.

Studies of the effects of acute doses of nicotine on behavioral measures of activity in animals indicate that nicotine may reduce negative affect in a number of different species (Bell, Warburton, Brown 1985; Emley and Hutchinson 1983). However, close inspection of the procedures used in these studies reveals that doses that suppress behavioral indices of emotion also may produce nicotine toxicity. Such high doses may decrease a large variety of behavioral indices due to the induction of physical distress. However, Silverman (1971), using doses of nicotine comparable to smoking doses, reported nicotine-induced reductions of aggression. Careful evaluation of studies of the effects of nicotine on indices of emotion in nonhuman subjects indicates that while these studies generally support the view that nicotine has inherent negative-affect-reducing properties independent of withdrawal effects, most have administered such high doses of nicotine as to make their relevance to habitual nicotine use in humans questionable.

Overall, evidence from experimental studies supports survey findings suggesting that tobacco use and nicotine consumption are associated with decreases in negative affect in habitual tobacco users. As was true for the learning and performance literature, caution must be exercised in generalizing about smoking and nicotine's effects on stress and mood because most laboratory studies compare smokers smoking with smokers not smoking. Few studies include the important control group of nonsmokers not smoking to allow unequivocal determinations of whether smoking and nicotine are stress reducing or whether smoking abstinence and nicotine deprivation are stress increasing. Certainly, it seems that smoking by smokers is stress reducing compared with smokers not smoking. The experimental literature suggests that smoking and nicotine may reduce negative affect most effectively in situations involving mild or moderate distal (anticipatory) anxiety and/or ambiguous stressors. The roles that individual differences in personality, temperament, and psychopathology may play in determining the nature or degree of the stress-reducing effects of nicotine are yet to be determined.

Suggested Mechanisms Underlying Nicotine's Effects on Stress and Mood

Based on the extant epidemiological literature linking stress and smoking and the laboratory studies indicating that stress increases smoking, several investigators have offered mechanisms to explain these relationships. These theoretical positions are varied and none has yet received unequivocal support to the exclusion of the other proposed mechanisms. Perhaps several or all of these mechanisms are operating. The major positions are reviewed below.

An Emphasis on Nicotine Withdrawal Symptoms

Schachter (1979) suggested that nicotine reduces negative affect in smokers simply by reducing symptoms of nicotine withdrawal. Increased irritability, anxiety, and depression are the most common symptoms of smoking withdrawal (Murray and Lawrence 1984), and these are the very emotions that appear to be most consistently reduced by acute doses of nicotine in nicotine-deprived smokers (Gilbert and Welser, in press). Thus, alleviation of withdrawal symptoms may account for the capacity of nicotine to reduce negative affect in nicotine-deprived smokers.

The degree to which an individual is physically dependent on nicotine may account for the variable effects observed. Perlick (1977) found that normal-nicotine-delivery cigarettes alleviated annoyance in heavy but not light smokers. On the other hand, the reduction in negative affect following nicotine administration may not be simply and solely a consequence of withdrawal symptom relief, because several investigations showing such effects used minimally deprived individuals who had not developed withdrawal symptoms (Pomerleau 1981).

A variant of this proposed mechanism suggests that smoking increases under stress and in dysphoric mood states because biological and psychological effects of stress and dysphoric moods are similar to the experience of nicotine withdrawal. From past experience, smokers learn that smoking alleviates these unpleasant states. Therefore, stressors and dysphoric moods come to elicit smoking because of conditioned responses or because of misattribution of the unpleasant experiences to nicotine withdrawal (Barefoot and Girodo 1972; Grunberg and Baum 1985). This misattribution model has some empirical support but requires careful examination.

Neurochemical Models

Evidence has been offered in support of the hypothesis that nicotine-induced release of glucocorticoids and other neuromodulators, such as the endogenous opioid beta-endorphin, may account for nicotine's capacity to reduce stress and negative affect (Gilbert 1979;

Pomerleau and Pomerleau 1984). While high doses of nicotine and rapid smoking of cigarettes after a period of smoking deprivation cause reliable increases in plasma concentrations of such neuromodulators (Seyler et al. 1986), it is not clear whether normal smoking during nonstressful conditions causes increases in these neuromodulators (Gilbert and Welser, in press). However, normal smoking in combination with mild-to-moderate stress may result in such increases. In addition, even if such neurochemical changes occur, it is not clear whether they act to modulate stress or dysphoric moods.

Biphasic Action on the Sympathetic Nervous System

Studies of human performance show that performance on simple tasks is improved by higher arousal, but performance on complex tasks is impaired by a high arousal level (Levine, Kramer, Levine 1975). In coping with the varying demands of daily life, at times it may be advantageous to vary the level of sympathetic nervous system (SNS) arousal. The ability to regulate arousal in this fashion would enable individuals to appraise stressful situations as less threatening and could result in improved performance in various conditions. There is some evidence suggesting that nicotine may have biphasic effects on SNS responses, producing either stimulatory effects or dampening effects under different conditions. Under conditions of low environmental demand, the effect of nicotine is generally to produce stimulatory or SNS arousal effects, including increases in heart rate and blood pressure (Grunberg and Baum 1985; MacDougall et al. 1983, 1986). This effect may be responsible for the perceived functions of "stimulation" or coping with "inactivity/boredom" (Best and Hakstian 1978; Coan 1973; Ikard, Green, Horn 1969; Leventhal and Avis 1976), and there is evidence indicating that smoking improves performance on simple tasks (Suraway and Cox 1986; Wesnes and Warburton 1983). At high levels of arousal, however, there is some evidence that nicotine produces central nervous system (CNS) tranquilization effects or reduces reactivity to stressful stimulation (Armitage, Hall, Sellers 1969; Ashton et al. 1974; Golding and Mangan 1982; Woodson et al. 1986). Evidence suggests that nicotine can restore high brain activation to moderate levels. In low-arousal situations, such as vigilance tasks, nicotine produces cortical activation and increased alertness (Edwards et al. 1985). Increased cortical activation could increase hedonic tone directly or indirectly by allowing the individual to perform more effectively on desired tasks and thus to experience indirect rewards such as the perception of increased self-efficacy. In contrast, nicotine has been associated with decreased cortical activation and reduced anxiety in stressful conditions (Gilbert 1985; Golding and Mangan 1982). Nicotine administration by smoking and

other means may allow individuals to achieve a hedonically more desirable level of cortical activation (Eysenck 1972).

At present, there is no direct evidence linking these physiological effects to perceived stress reduction or improved performance under stressful conditions. This position is also consistent with the findings reported in the first Section of this Chapter.

Altered Body Activity

Several mechanisms based on altered body activity may account for nicotine's stress-reducing effects. First, based on evidence that nicotine may in some situations increase the threshold for electric shock (Mendenhall 1925) and on the observation that nicotine-induced increases in cardiovascular activity typically do not produce corresponding increases in perceived heart activity (Gilbert and Hagen 1980), nicotine may reduce the intensity of emotional experiences by increasing perceptual thresholds for emotion-related feelings of bodily arousal (Gilbert 1979). The small number of studies evaluating this hypothesis have provided mixed results (Sult and Moss 1986), possibly because some have not been carried out under conditions of high stress. This elevated perceptual threshold model is consistent with the CNS arousal modulation model and with the neuromodulator model in predicting that under conditions of heightened stress, nicotine should elevate perceptual and pain-endurance thresholds.

A related possibility is that smoking reduces sensitivity to painful stimuli and sensitivity to internal proprioceptive cues that produce discomfort. Antinociceptive action (i.e., reducing perception of pain stimuli) has been documented in several animal studies (Friedman, Horvath, Meares 1974; Sahley and Berntson 1979; Tripathi, Martin, Aceto 1982). Evidence from humans is mixed, with several studies showing that smoking increases tolerance to painful stimuli (Pomerleau, Turk, Fertig 1984; Nesbitt 1973; Silverstein 1982), and the effect is attributable specifically to nicotine intake rather than to the physical act of smoking (Fertig, Pomerleau, Sanders 1986). Several studies have failed to find effects of smoking on pain thresholds (Shiffman and Jarvik 1984; Sult and Moss 1986; Waller et al. 1983). These null results may be attributable to methodological details such as gender differences or differences in current nicotine level.

Another possibility is that nicotine produces a state of tranquillity or relaxation by reducing the level of tonic and/or phasic muscular activity (Gilbert 1979). Experimental evidence strongly supports the view that nicotine depresses certain muscular reflexes (Domino 1979; Hutchinson and Emley 1973). Ginzel and Eldred (1972) and Ginzel (1987) have shown that nicotine produces muscle relaxation in the cat. Epstein and coworkers (1984) have reported that smoking by humans reduces sensitivity to perception of muscle tension.

Schachter (1973) suggests that nicotine reduces emotional experience by reducing emotion-induced phasic increases in autonomic nervous system (ANS) activity. Because nicotine typically increases activation of the ANS, this increase in tonic ANS activation should produce a ceiling effect such that the additional arousal increase associated with the onset of emotional stimulation is less than the emotion-induced arousal that occurs without nicotine. This third hypothesis assumes that phasic, rather than tonic, activation of the ANS is an important contributor to the subjective experience of emotion. Consistent with this possibility, nicotine increases tonic heart rate, but reduces phasic heart rate responses to stressors (Schachter 1973; Woodson et al. 1986).

Hedonic Systems Model

Nicotine-induced modulation of one or more systems in the brain associated with pain and pleasure may account for the capacity of nicotine to reduce negative affect and increase feeling of well-being (Eysenck 1973; Jarvik 1973). Eysenck (1973) suggests that feelings of well-being produced by nicotine and other means can be increased by influencing three hedonic systems: the primary reward, the primary aversion, and the secondary reward systems. Activating the primary system is thought to produce pleasure directly, while activating the secondary reward system produces rewarding effects indirectly, by inhibiting the aversion system. Eysenck suggests that nicotine administered during highly stressful situations may improve mood by means of the secondary system, while nicotine administered during low-arousal conditions may directly stimulate primary reward systems. Any primary rewarding effect of nicotine appears to be very subtle; many smokers and a smaller percentage of nonsmokers report pleasurable stimulant effects following the administration of nicotine (Jones, Farrell, Herning 1978). However, the subjective effects of nicotine appear to depend greatly upon expectations (Hughes et al. 1985); individuals who are not habitual tobacco users typically report that nicotine administered in any form produces unpleasant effects (Nyberg et al. 1982). In addition, the biochemical representation of affective states is not well understood (McNeal and Cimbolic 1986), and these states are a joint function of physiological and psychological factors (Reisenzein 1983; Schachter and Singer 1962). Experimental studies of stressful situations have shown that smoking produces reduction in subjective ratings of anxiety (Jarvik et al., in press; Pomerleau, Turk, Fertig 1984), but several studies have failed to find effects of smoking for subjective anxiety (Fleming and Lombardo 1987; Shiffman and Jarvik 1984) or emotional behavior (Hatch, Bierner, Fisher 1983). It appears that anxiety-reduction effects are observed primarily when smoking occurs before, rather than during, the stressful situation (Gilbert, in press).

Therefore, the anxiety reduction may result from cognitive appraisal rather than from direct reduction of negative affect, but it should be noted that comparable patterns of findings are commonly observed for most anxiolytic medications (Janke 1983).

Regarding positive affect, it has been suggested that effects of nicotine on endogenous opioid systems may relate to experienced pleasure (Pomerleau and Pomerleau 1984). There is some evidence that effects of cigarette smoke on the upper and lower respiratory airways contribute to pleasurable functions of smoking (Rose et al. 1985), but direct evidence of an influence on positive affect has not been demonstrated.

Lateralized Affective Processors Model

The capacity of nicotine to decrease negative affect may stem from its capacity to increase activation of the left cerebral hemisphere compared with the right hemisphere (Gilbert 1985). Lateralized effects on electrocortical (Elbert and Birbaumer 1987; Gilbert 1985; Gilbert, in press) and electrodermal (Boyd and Maltzman 1984) activity have been reported. These electrophysiological studies along with behavioral studies (Gilbert and Welser, in press) suggest that during stressful/high-arousal conditions, nicotine reduces right-hemisphere more than left-hemisphere parietal activation, while during low-stress situations it may activate the right hemisphere more than the left. Activation of the right hemisphere appears to be more related to the experience of negative affect (Davidson 1984), while the left hemisphere is more the biological seat of logical sequential and verbal information processing (Tucker and Williamson 1984). Thus, nicotine-induced reductions of right-hemisphere activation are associated with reductions in negative affect. Consistent with this finding, simultaneous reductions in right-hemisphere EEG activation and in negative affect have been reported while subjects viewed a stressful movie (Gilbert 1985). These lateralized effects may occur as a result of nicotine's influence on one or more relatively lateralized neurotransmitter systems (Gilbert and Hagen 1980). The lateralized effect model suggests a common biological basis for a diverse set of psychological and physiological effects of nicotine.

Hypothalamic Consummatory Drive Model

Both exposure to nicotine and the activity of the hypothalamus are linked to hunger and body weight, as well as to affective, cognitive, and perceptual processes. Stimulation of the ventromedial hypothalamus or deactivation of the dorsolateral hypothalamus produces effects similar to those produced by the administration of nicotine: decreased emotionality, decreased sensitivity to distracting stimuli,

heightened activity level, low taste responsivity, and weight loss (Nisbett 1972). Nicotine withdrawal, as well as lesions of the ventromedial hypothalamus or stimulation of the dorsolateral hypothalamus (Nisbett 1972), leads to the opposite effects: increased emotionality, increased distraction by external stimuli, decreased activity level, increased taste responsivity, and weight gain (Grunberg and Baum 1985; Perlick 1977). There are a number of commonalities between nicotine and food consumption (Grunberg and Baum 1985). Food consumption, like nicotine, reduces anxiety (Schachter 1971), and many individuals smoke (Rose, Ananda, Jarvik 1983) and/or eat more (Morley, Levine, Rowland 1983) when anxious. Nicotine may reduce aspects of the hunger drive (Grunberg and Baum 1985) and may be reinforcing for this reason. The hypothalamic consummatory drive model suggests that consummatory drive reduction by nicotine should reduce the agitation and irritability associated with a high drive state.

Indirect Models: Psychological Enhancement and Sensory Gratification

Nicotine may reduce negative affect indirectly by enhancing cognitive functioning and associated task performance (Ashton and Stepney 1982; Wesnes and Warburton 1978). The effects of smoking and nicotine on performance (reviewed earlier in this Chapter) are consistent with this interpretation. Nicotine may improve affect both directly, by one or several of the mechanisms discussed above, and indirectly, by enhancing certain psychological processes. Moreover, there is evidence that smoking improves visual sensory processing while blunting auditory distractors in humans (Friedman and Meares 1980).

Sensory experiences related to tobacco consumption may contribute to the motivation for its use and its affect and stress-related effects. Some smokers report smoking because they like handling cigarettes, watching smoke, and/or the sensory experience of smoke in the throat and lungs (Russell, Peto, Patel 1974). Experimental studies, although limited in number, have supported the view that sensory factors are important contributors to the satisfaction and craving-reduction associated with smoking (Rose et al. 1985). The strong sensory impact associated with all forms of common tobacco use may also reduce negative affect by providing distraction from negative thoughts and stimulation that relieves boredom (Gilbert and Welser, in press).

Implications for Tobacco Use

Stress is a risk factor for smoking initiation and increases cigarette smoking (e.g., puffs per cigarette) among regular users.

Smoking is stress reducing for many smokers, and nicotine appears to be involved in this effect. It is likely that the effects of nicotine on stress and on mood involve several mechanisms including alleviation of withdrawal symptoms, peripheral muscle relaxation, central neurochemical changes and electrocortical arousal, interaction with consummatory reward systems, and indirect effects such as psychological enhancement and sensory gratification. Future research needs to address and compare the possible mechanisms. Regardless of which mechanisms are operating, the relationship between stress and smoking undoubtedly reinforces habitual tobacco use and may contribute to initiation and relapse.

Tobacco Use, Nicotine, and Body Weight

Cigarette smokers weigh less than comparably aged nonsmokers, and many smokers who quit smoking gain weight (Grunberg 1986a; Rodin and Wack 1984; Wack and Rodin 1982). It has been suggested that some people smoke to prevent weight gain as the result of smoking cessation (Birch 1975; Charlton 1984b; Grunberg 1986a). Therefore, methods to control weight gain following cessation have been recommended (Birch 1975; Ducimetiere et al. 1978; Grinstead 1981; Grunberg and Bowen 1985a). How much weight gain actually occurs following smoking cessation (Albanes et al. 1987; Bosse, Garvey, Costa 1980; Rabkin 1984; Wack and Rodin 1982), the specific mechanisms (i.e., changes in dietary intake, physical activity, and/or changes in resting metabolic rate) responsible for this weight gain (Grunberg 1986b; Hofstetter et al. 1986), and whether weight gain (or fear of weight gain) affects either cessation or relapse efforts (Hall, Ginsberg, Jones 1986; Klesges and Klesges, in press; Kramer 1982) remain controversial. This Section reviews data relevant to the smoking/body weight relationship.

The Relationship Between Smoking and Body Weight

The relationship between smoking and body weight has been extensively examined and reported for more than 100 years (Kitchen 1889; Otis 1884). Human studies can be summarized into two broad areas: (1) cross-sectional evaluations that have compared the weights of smokers, nonsmokers, and in some cases, ex-smokers; and (2) longitudinal, within-subject evaluations that have measured weight changes in smokers, ex-smokers, and nonsmokers over time. The cross-sectional evaluations reported since 1970 are tabulated in Table 2, and the longitudinal studies reported since 1970 are summarized in Table 3. Both tables present the reference and year, a brief description of the sample design, major findings, observed moderator variables (e.g., gender, number of cigarettes per day) for weight, and major limitations of the study. Only studies published

since 1970 are summarized in this Report because there are so many studies and because reviews of earlier investigations (Bosse, Garvey, Costa 1980; Grunberg 1986a) indicate that the results are completely consistent with the studies presented in Tables 2 and 3.

Cross-Sectional Evaluations of Smoking and Body Weight

Of the 28 cross-sectional evaluations presented in Table 2, 25 (89 percent) reported that smokers weigh less than nonsmokers. An additional study (Sutherland et al. 1980) found this relationship for women but not for men and another study (Hjermann et al. 1976) found this relationship for older (45 to 49 years) but not younger (40 to 44 years) men. Only one study did not report an inverse relationship between smoking and body weight, and that study examined visitors to a "health exhibit," a population that may be health conscious and predisposed to making positive health changes (Waller and Brooks 1972). This one discrepant study included a high percentage of cigar and pipe smokers (many of whom do not inhale). While it is difficult to summarize the cross-sectional studies because of differences in reporting techniques, it was found that smokers overall weighed an average of 7.13 lb (range: 2.36 to 14.99) less than nonsmokers.

Because smoking and alcohol consumption are correlated, one study (Williamson et al. 1987) examined, through multivariate methods, the effects of smoking and alcohol consumption on body weight. This study reported that alcohol consumption accounted for approximately 44 percent of the reduction in body weight in women who smoked compared with women who did not smoke. For men, statistical adjustment for alcohol consumption did not alter the weight-lowering effect of smoking.

Cigarette consumption, age, and gender have been adequately evaluated to reach some conclusions regarding their impact on the relationship between smoking and body weight. The effect of cigarette consumption has been parametrically evaluated in eight studies. Six (Albanes et al. 1987; Hjermann 1976; Holcomb and Meigs 1972; Jacobs and Gottenborg 1981; Khosla and Lowe 1971; Lincoln 1970; Stephens and Pederson 1983) of the eight investigations (75 percent) reported a nonlinear relationship. In all of these reports, nonsmokers had the greatest body weights; moderate smokers (typically 10 to 20 cigarettes/day) had the lowest body weights; and some heavy smokers (typically >20 cigarettes/day) had body weights approaching that of nonsmokers. Two studies (Bjelke 1971; Kopczynski 1972) reported no relationship between level of smoking and weight.

The effect of age on the smoking/body weight relationship was examined in six investigations. Five of six studies (86 percent) (Albanes et al. 1987; Bjelke 1971; Hjermann et al. 1976; Jacobs and

TABLE 2.—Cross-sectional evaluations of smoking and body weight

Study	Design and sample	Major results	Moderator variables	Limitations
Albanes et al. (1987)	12,103 men and women, NHANES II Survey	Smokers weighed 5.95 lb less than nonsmokers, controlled for age, sex; smokers taller and leaner than nonsmokers, based on skinfold	Age: current smokers gained less after age 25 than either nonsmokers or ex-smokers Smoking duration: body mass index decreased with smoking duration increase Smoking rate: moderate smokers leaner than low or high rate smokers	Smoking self-report
Andrews and McGarry (1972)	All 18,631 pregnant women, Cardiff, Wales, 1965–1968	Across all heights, smoking mothers lighter than nonsmokers		Pregnant women only; birth survey record data; actual weight changes not presented
Biener (1981)	274 (174 men, 100 women) ex-smokers, worksite setting	49% women, 39% men gained weight following cessation; quitter approximate average gain: women 11 lb, men 15 lb		Retrospective postcessation gain self-report; no nonsmoker control group
Blair et al. (1980)	183 white male, 284 white female insurance company employees; average age 34	Smokers 2.64–7.5 lb lighter than nonsmokers, 0.88–15.21 lb lighter than ex-smokers; smaller skinfolds for smokers of both sexes than nonsmokers		Small sample size; white office workers only

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Bjelke (1971)	8,638 male, 10,331 female respondents, mail survey, Norway general population "systematic sample"	Used "bulk index" (weight/height^2); both sexes current smokers less bulky than quitters and never smokers	Smoking rate: not related to weight Age: older respondents greater smoker/nonsmoker bulk differences Sex: women greater smoker/nonsmoker bulk differences	Self-report by mail; no weights, no statistical analyses presented
Fehily et al. (1984)	211 nonsmoking, 282 smoking men, aged 45–59, heart disease study	Smokers weighed 7.5–10.3 lb less than nonsmokers, 6.6–9.4 lb less than ex-smokers; pipe/cigar smokers weighed 2.4 lb more than nonsmokers; weight/height^2 index results similar		Small, all white, restricted sample; smoking self-report
Fisher and Gordon (1985)	15% random sample, 10 U.S., Canadian clinics; 2,269 male, 2,105 female whites, aged 20–59, LRC Prevalence Study	Men: smoking nondrinkers weighed 6.6 lb less than nonsmoking nondrinkers; smoking drinkers weighed 2.2 lb less than nonsmoking drinkers Women: smoking nondrinkers weighed 2.2 lb less than nonsmoking nondrinkers; smoking drinkers weighed 4.4 lb less than nonsmoking drinkers		All white population; smoking self-report
Friedman et al. (1981)	38 smoking-discordant monozygotic twin pairs, average age 40 years	Smokers weighed 5.07 lb less than nonsmokers		Self-report by mail; small restricted sample

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Garn et al. (1978b)	17,649 pregnant women, national health survey	Smoking mothers prepregnancy weight less than nonsmoking mothers; difference: whites 2.43 lb, blacks 3.53 lb	SES and race: no smoking/weight relationship influence	Pregnant women only; self-reports
Garrison et al. (1983)	Framingham study participants; assessed 1949–1952	Nonsmokers 55% of highest weight group; smokers 80% of lowest weight group		Sample size, weights not given; no statistical evaluation
Goldbourt and Medalie (1977)	10,059 male government workers, aged 40–65	Current smokers 1/4 inch taller, 2.36 lb less than nonsmokers; ex-smokers in between; leaner skinfolds for smokers than ex-smokers and nonsmokers		Limited age range, employment group; smoking self-report
Gyntelberg and Meyer (1974)	5,249 employed men, aged 40–59, Denmark	Nondrinking smokers 1.5 percentile points lighter than nondrinking nonsmokers; light drinking smokers 2.9 percentile points lighter; heavy drinking smokers 5.9 percentile points lighter than drinking nonsmokers		All-male sample, one city; smoking self-report
Hjermann et al. (1976)	Approximately 18,000 male participants, aged 40–49, coronary risk factor screening, Oslo	Aged 45–49 smokers body weight 3.09 lb less than nonsmokers; aged 40–44 difference not significant; no group weight/height^2 index differences	Smoking rate: heavy smoker (>20/day) body weights higher than lighter smoker Age: older smokers (45–49) weighed less than nonsmokers; younger smokers (40–44) no effect	Smoking self-report; limited age range; one city; all men

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Holcomb and Meigs (1972)	226 manufacturing company male hourly employees, aged 55–59	Mild to moderate smokers 14 lb lighter than never smokers, ex-smokers, and heavy smokers	Smoking rate: heavy smokers (>1 pack/day) heavier than lighter smokers, equivalent to nonsmokers	Smoking self-report; limited age, incomes; all men
Huston and Stenson (1974)	184 men, British Field Regiment	≤10 mm subscapular skinfold men averaged 22 cigarettes/day; ≥15 mm subscapular skinfold men averaged 12 cigarettes/day		Limited male sample; smoking self-report; no separate smoker/ nonsmoker data
Jacobs and Gottenborg (1981)	3,291 white men and women, aged 20–59, no cardiovascular disease or elevated risk factors; randomly selected middle-class suburb census tract blacks	Smokers lighter than never smokers and quitters	Smoking rate: male moderate smokers (14–29 cigarettes/day) 6.39 lb lighter than nonsmokers, 2.65–9.93 lb lighter than light and heavy smokers; female moderate smokers 5.07 lb lighter than never smokers, 1.54–8.38 lb lighter than heavy smokers Age: moderate/never smoker weight difference increased with age	Smoking self-report; restricted population
Khosla and Lowe (1971)	10,482 male steel workers, Wales	Per weight/height^2 index, smokers lighter than nonsmokers	Smoking rate: heavy smokers (>35 cigarettes/day) heavier than moderate smokers (15–34) Age: group weight differences increased after age 35	Smoking self-report; restricted population
Kittel et al. (1978)	8,284 male factory workers, Belgium	Relative weights significantly lower for cigarette smokers than never smokers, ex-smokers, and pipe/cigar smokers		Limited population, risk factor Rx program

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Kopczynski (1972)	3,059 random selectees, pulmonary disease study, Poland	Nonsmokers heavier than smokers, except 20-year-old men	Sex, age, smoking rate: no smoking/weight relationship influence	Smoking self-report; weights not reported
Lincoln (1970)	3,220 male household heads, aged 41–70, across United States	Smokers weighed 3–14 lb less than nonsmokers	SES: smoker/nonsmoker weight difference increased as income decreased Smoking rate: heavy smokers (≥21 cigarettes/day) weighed 4 lb more, moderate smokers (11–20 cigarettes/day) 4 lb less than all-smoker average	Restricted population; men
Matsuya (1982)	90 telephone employees, Japan	Ex-smokers weighed 5.29 lb more than nonsmokers; light smokers 2.87 lb less, heavy smokers 0.44 lb less than ex-smokers		Small, nonrepresentative sample; data self-report
Nemery et al. (1983)	210 steelworkers, aged 45–55, >10 years' service, Belgium	Smokers weighed 12.13 lb less than never smokers, 14.33 lb less than ex-smokers		Restricted population; smoking self-report
Stamford et al. (1984a)	164 (56 smokers, 108 nonsmokers) premenopausal women; smokers: ≥20 cigarettes/day, ≥5 years, inhale	Smokers weighed 11.96 lb less, had lower average Quetelet Index than nonsmokers		Small sample size; premenopausal women only; data self-report
Stamford et al. (1984b)	269 adult men, fitness center screened; smokers: ≥20 cigarettes/day, ≥5 years, inhale	Smokers weighed 14.99 lb less, had 12% less body fat than nonsmokers		Select sample, exercising men; smoking self-report; heavy smokers

TABLE 2.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Stephens and Pederson (1983)	15,518 persons aged >10; questionnaire, anthropometry	Smokers weighed less than nonsmokers; female smokers weighed 1.32 lb more to 5.73 lb less than female nonsmokers; men weighed 3.09–7.7 lb less; smokers averaged 3.445 lb less than nonsmokers		White women self-report, smoking self-report; no statistical significance tests
Sutherland et al. (1980)	Random sample, 175 men and women, rural town, New Zealand	Weight/height^2 index and skinfolds significantly higher in nonsmoking than smoking women; higher for nonsmoking men, but not significant	Sex: male smokers not significantly leaner than nonsmokers; smoking women lighter than nonsmoking women	Smoking self-report; small sample size
Waller and Brooks (1972)	2,169 health exhibit visitors	"Little weight difference" among current smokers, nonsmokers, and ex-smokers		Smoking self-report; bathroom scale weight; health-conscious population; high % cigar/pipe smokers; no statistical evaluations
Zeiner-Henriksen (1976)	Approximately 15,000 randomly selected Norwegians	Current smokers average and relative weight lower than nonsmokers or ex-smokers		Smoking and weight self-report, questionnaire

TABLE 3.—Longitudinal evaluations of smoking and body weight

Study	Design and sample	Major results	Moderator variables	Limitations
Blitzer et al. (1977)	57,032 women, aged 20–59, self-help weight loss groups	Quitters gained 7.0–10.2 lb more than continuing smokers	Smoking rate: weight gain/previous smoking rate proportional	Smoking and weight self-reports; all women trying to lose weight
Bosse et al. (1980)	1,749 adult men, Normative Aging Study, assessed over 5 years	Average 5-year gains: never smokers 1.81 lb; former smokers 1.87 lb; current smokers 2.00 lb; ex-smokers who quit 6.34 lb	Age: younger quitters gained more Adiposity: fatter quitters gained more Tar rate: higher pretest tar rate smokers gained most Anxiety: high related to higher gain	Smoking self-reports; all men; actual weights not presented
Burse et al. (1982)	4 paid volunteers; 11-day baseline, 21-day quit period, 20-day resumption period	3 of 4 gained weight; 1.98 lb increase during cessation; 1.76 lb loss on resumption		Very small sample, paid volunteers; short-term evaluation
Cambien et al. (1981)	1,097 Paris civil servants, aged 25–35, screened, randomly assigned, cardiovascular risk factor reduction intervention or control groups; 2-year followup evaluation	Treatment group quitters gained 4.85 lb, control group quitters 7.50 lb; nonsmokers and no-change smokers gained 1.54 lb in treatment group, 2.2 lb in control		Smoking self-report; risk factor reduction program participants
Carney and Goldberg (1984)	13 women, 5 men, aged 28–67, smoked $\geq$20 cigarettes/day, $\geq$5 years; 12 male controls; 15 smokers abstained 2 weeks	Quitters weight change range: −3.09 to +9.0 lb	Smoking rate/duration: no weight change relationship Biological variables: weight gain positively related to lipoprotein lipase activity in adipose tissue	Smoking self-report; controls weight changes not reported; short-term evaluation

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Coates and Li (1983)	373 male asbestos-exposed smokers, aged ≥42; 87% white, mean education 12.8 years; 12 months assessment after cessation effort	Continuous quitters gained 5.15 lb; continuous smokers gained 0.35 lb		Smoking self-report; all male, nonrandom sample
Comstock and Stone (1972)	502 male telephone workers, aged 40–59, mostly white; 2 assessments 5 years apart	5-year followup average gains: never smokers 2.43 lb, ex-smokers 5.07 lb, continuing smokers 2.42 lb; quitters 11.24 lb and showed greatest skinfold *increases*	Smoking rate: increasing quitter weight gain with heavier prequit smoking	Smoking self-report; men only
Dallosso and James (1984)	16 (8 men, 8 women) antismoking clinic participants; mean age, men 47.1, women 35.4; assessed before and 6 weeks after clinic	10 quitters gained 3.00 lb; 5 continuing smokers lost 0.99 lb		Small sample size; smoking self-report; limited followup
Emont and Cummings (1987)	125 stop-smoking clinic participants; pretreatment and 1-month followup assessments	76% quitters and slippers (≤5 cigarettes/day) averaged 5.8 lb gain	Nicotine gum: gain/gum use reliable negative correlation for heavy smokers: gain not related to age, sex, marital status, baseline body weight	Weight gain, smoking self-report, confounded by gum use; limited followup; incomplete data
Fagerström (1987)	28 nicotine gum users; abstinent at 6 months	Infrequent gum users gained 6.83 lb, frequent users 1.98 lb	Nicotine gum: frequent users gained less weight	Small sample size; measures unclear
Friedman and Siegelaub (1980)	Multiphasic health checkup patients; smoked, then quit 12–18 months later (N=3,825) or continued (N=9,392)	Quitters gained 2–3 lb more than continuing smokers	Smoking rate: higher initial smoking rate related to greater weight gain after cessation	Smoking self-report; whites only data

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Garn et al. (1978b)	6,979 women followed through ≥2 pregnancies	Higher prepregnancy weights for habitual nonsmokers than habitual smokers: whites 3.4 lb, blacks 4.1 lb; lower habitual smoker gains between pregnancies for both races	Race: no weight/smoking relationship influence	Smoking self-reports; restricted population
Garvey et al. (1974)	870 white male veterans, aging study, assessed 4–7 years after initial assessment	Smoking/weight change significantly related; recent quitters (≤5 years) gained 4.19 lb more than smokers, nonsmokers, former smokers	Age: 40–54 quitter weight increase greatest	Smoking self-report; exact quit date unknown
Glauser et al. (1970)	7 male smokers, cessation program; assessed preprogram, 1 month postprogram	At 1-month followup, participants gained 6.4 lb		Smoking self-report; exact quit date unknown
Gordon et al. (1975)	4,798 Framingham study participants: 1,498 male smokers, 492 male nonsmokers, 1,634 female nonsmokers, 1,174 female smokers; examined short-term changes after biennial exam 1, long-term effects between biennial exams 4, 10	At entry, male smokers weighed 8.0 lb less than nonsmokers; short-term male quitters gained 3.8 lb, nonsmokers 0.5 lb, continuing smokers 0.3 lb; new smokers lost 9 lb; too few female quitters to evaluate		Smoking self-report; change analysis, men only
Gormican et al. (1980)	301 pregnancy obstetrics records, women, aged 17–35	Smoker, nonsmoker prepregnancy weight similar; no last 2 trimester weight gain difference (nonsmokers 24.6 lb, smokers 22.6 lb)		Clinic record data; pregnancy weight gain data only

TABLE 3.—Continued

Study	Design and sample	Major results	Moderator variables	Limitations
Grinstead (1981)	45 subjects (38 women, 7 men), average age 40; evaluated 6 months after cessation treatment; saliva thiocyanate verification	During program, 63% subjects averaged 2.88 lb increase, 34% averaged 2.46 lb decrease; at followup, 37% averaged 6.97 lb gain, 43% averaged 3.27 lb loss		Questionnaire, phone interview data
Gritz et al. (in press)	554 self-quitters (245 men, 309 women), mean age 41.4, 85% Caucasian, 9% black, 4% Asian, 1% Asian-American, 1% Native American; 1-year followup	35% previous quitters gained, 3% lost; at 1 year, abstainers averaged 6.1 lb gain; relapsers gained 2.71 lb while abstinent, lost 1.3 lb upon relapse; continuous smokers gained 0.3 lb		Questionnaire, phone interview data
Grossarth-Maticek et al. (1983)	1,353 subjects, Yugoslavian village of 14,000; every 2d household oldest member; evaluated 1965–1966, 1969	Smoking reduction/weight increase relationship (regression coefficient -0.30)		Smoking self-report; weights, weight changes not reported
Gunn and Shapiro (1985)	89 cessation clinic participants; all quit at initial evaluation; 3-month followup assessment	43 of 54 (80%) quitters gained 2–30 lb		Smoking, height, weight self-report; inadequate statistical evaluation
Hall et al. (1986)	255 smoker participants (122 men, 133 women), 2 smoking treatment trials; 6-, 12-month followups; biochemical verification	Abstainers gained more than smokers at 1 year	Smoking rate: pretest smoking level/postcessation weight gain positively related Chronic dieting: chronic diet subjects gained most	Multiple Rx (e.g., nicotine gum) participant data included
Hatsukami et al. (1984)	27 smokers hospitalized 7 days; 20 subjects smoked 3 days, then quit 4 days; 7 control group subjects smoked throughout	Quitters gained 1.76 lb in 4 days		Small sample size; inpatient environment